usual site of gastric ulcer. Further, almost every case gives a clinical history suggesting gastric ulcer for a long period of years preceding the relatively short period when the history became that of gastric cancer.

That carcinomas should develop in the edges of gastric ulcers is only what we should expect; the wonder is that the facts should have been so long in being recognized. This has been due to: (*a*) Failure to recognize clinically the frequency of gastric ulcer; (*b*) failure to recognize that gastric cancers are not initially pyloric tumors, but extensions thereto from the lesser curvature; and (*c*) giving undue weight to observations at autopsies. When the patient has died of gastric cancer, the neoplasm has usually obliterated all gross and microscopic evidence of previous ulcer.

As the pathologist examines stomach specimens from the surgical clinic he constantly observes the various steps in the following sequence:

1. Chronic ulcers from the centres of which the mucosa has disappeared leaving a scar tissue base.
2. In the overhanging broders of the ulcers the mucosa is proliferating.
3. Deep in the borders many groups of epithelial cells have been nipped off by scar tissue and are exhibiting all stages of aberrant proliferation with infiltration of the surrounding tissues.
4. Metastases are forming in the lymphatics of the stomach wall and adnexa.

A small percentage of cases operated upon are too far advanced to show these steps, and a very small percentage—probably not over 2 per cent.—give evidence of rapid aberrant epithelial proliferation and infiltration without any sign of previous ulcer.

Adopting Adami's[5] classification we may therefore correctly desig-designate most gastric carcinomas as "blastomas originating from unipotential cells of postnatal displacement," although it is probable that a very small number are "blastomas originating from unipotential cells that assume neoplastic characters without displacement and rapidly assume malignancy."

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[5] Principles of Pathology, 1908 i, 770.

THE ETIOLOGY AND PATHOLOGY OF INGUINAL HERNIA.

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IN order to understand the mode of occurrence of inguinal hernia and employ intelligent operative measures for its cure, the surgeon must be familiar with those structural conditions which make it possible for a portion of the intra-abdominal contents to protrude through the inguinal triangle, a space bounded above by a line drawn horizontally inward from the junction of the middle and outer thirds of Poupart's ligament to the outer margin of the rectus muscle, below by the inner two-thirds of Poupart's ligament, and internally by the outer margin of the rectus muscle. The structures to be considered are the inguinal fossæ, Hesselbach's triangle, the internal abdominal ring, the inguinal canal, and the external abdominal ring.

When the anterior abdominal wall is viewed from behind the following structures are seen: Extending from the apex of the bladder to the umbilicus is the urachus, the degenerated intra-embryonic portion of the allantois, also known as the median vesico-umbilical ligament. On either side of this median cord there is another, extending obliquely upward and inward to join it at the umbilicus. These are the remains of the hypogastric arteries, which are patent only during intra-uterine life. They are also called the lateral vesico-umbilical ligaments. Lying external to these lateral cords are the deep epigastric arteries. These five structures are covered with peritoneum, arranged in more or less well-defined folds, that covering each obliterated hypogastric and deep epigastric artery being known as the plica hypogastrica and plica epigastrica respectively.

In relation with these cords three depressions may be distinguished—one between the urachus and the obliterated hypogastric artery, another between the hypogastric and the epigastric arteries, and a third external to the epigastric artery. These depressions are known as the internal, middle, and external inguinal fossæ respectively, and with the exception of the internal one are very important with reference to the occurrence of inguinal hernia (Fig. 1). They can be examined better after the peritoneum has been carefully stripped off. When this has been done it will be found that the internal fossa is bounded in front, with the exception of a small area above its inferior external angle, by the rectus abdominis muscle, which is strong and unyielding, and well adapted to withstand any unusual intra-abdominal pressure which may be exerted upon it. Therefore, only the small area of this space which is not covered by the rectus muscle can be considered as a weak spot in the abdominal wall. It is quite different, however, with the middle fossa, the one between the obliterated hypogastric and the deep epigastric arteries. This one is bounded in front only by the transversalis fascia and the attenuated lower fibers of the internal oblique muscle. An examination of several hundred cadavers has shown that there is not a decided blending of the lower fibers of the internal oblique muscle with those of the transversalis to form a well-developed conjoined tendon in more than one-fourth of all cases. That portion of the internal oblique arising from Poupart's ligament has often been found to be aponeurotic instead of muscular, and to fuse almost imperceptibly with the

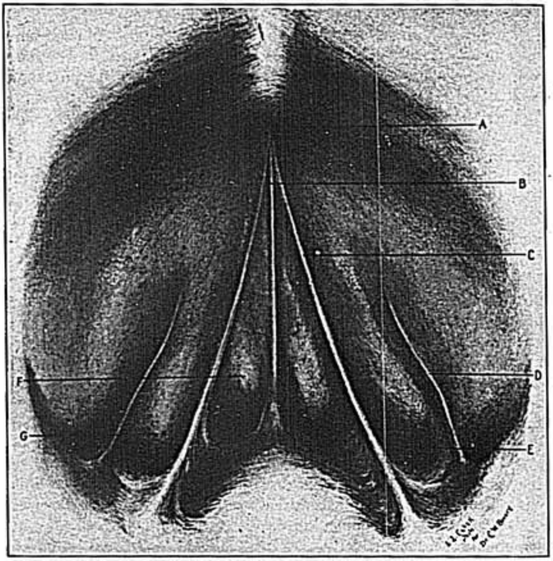

FIG. 1.—The anterior abdominal wall viewed from behind, showing the inguinal fossæ: *A*, umbilicus; *B*, urachus; *C*, obliterated hypogastric artery; *D*, deep epigastric artery; *E*, external inguinal fossa; *F*, internal inguinal fossa; *G*, middle inguinal fossa. (Drawing made from a dissection prepared by Dr. Bonney, Department of General Anatomy, Jefferson Medical College.)

aponeurosis of the transversalis; whether it stops short or is continued downward with the latter to help form the deep crural arch cannot positively be stated, for the reason that it has not been possible satisfactorily to dissect the one from the other so homogeneously are they united. Even when a decided tendinous union takes place there is always a considerable area below it which is filled in only by fascia. Thus, it is seen that the middle fossa is a decidedly weak area in the anterior abdominal wall, ill adapted to resist the force of any unusual intra-abdominal pressure which may be brought to bear upon it. In fact, it is through this area and the contiguous small uncovered portion of the internal inguinal fossa already described that a certain percentage of inguinal herniæ occur. The space thus formed, which is bounded externally by the deep epigastric artery, internally by the outer margin of the rectus muscle, and inferiorly by Poupart's ligament, is known as Hesselbach's triangle.

The external inguinal fossa, lying external to the deep epigastric artery, constitutes another weak spot in the anterior abdominal wall. This fossa really owes its existence to the internal abdominal ring, a spot in the transversalis fascia through which the testicle pushes its way on its course downward from the abdominal cavity to the scrotum. If the transversalis fascia be examined carefully at this spot after the peritoneum has been stripped away, the following arrangement will be found. Internally it is strengthened by tendinous fibers passing upward from the inner end of Poupart's ligament, and externally and superiorly it presents a somewhat annular arrangement of its fibers, which extend downward along the spermatic cord. To this funnel-shaped process, the term infundibuliform fascia is applied, and the internal stronger margin is known as the interfoveolar ligament of Hesselbach. The latter really forms a line of dermarcation between the external and middle fossæ just as the deep epigastric artery does (Fig. 2).

The formation of the internal ring and the inguinal canal is best made clear by tracing the descent of the testicle from the abdomen to the scrotum. The scrotum is formed from the genital swellings, into which a little sac of peritoneum is invaginated comparatively early in fœtal life. The inguinal ligaments, or gubernacula, are also attached to the bottom of this peritoneal pouch. Now as the fœtus develops, this pouch (which in the male is called the vaginal process and in the female the canal of Nuck) becomes more and more elongated, keeping pace with the growth of the genital swellings, until toward the termination of intra-uterine life, probably at about the end of the eighth month, it has formed a sac extending well down into the scrotum. It is to be borne in mind that this sac is placed in front of the testicle before that organ emerges from the abdomen, that it always bears such a relation to the testicle, and that it is not pushed from the anterior abdominal wall by the testicle in the way in which the other layers enveloping that gland and the spermatic cord are displaced. From these considerations it becomes evident that at a certain stage of fœtal life there is a free communication between the peritoneal cavity and the scrotum. Shortly after birth—probably between the tenth and twentieth days—the vaginal process normally becomes obliterated except as to its lower portion, which surrounds the testicle and forms its tunica vaginalis. Failure of the upper portion of this peritoneal process to become obliterated constitutes an important factor in the production of congenital hernia. Reference to it will be made again.

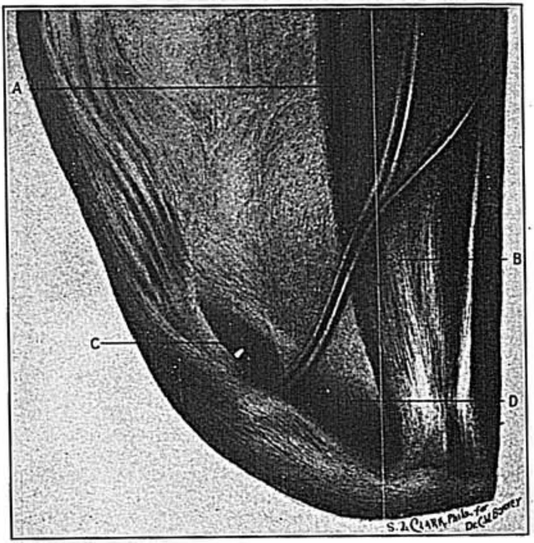

FIG. 2.—The anterior abdominal wall viewed from behind after the peritoneum has been stripped up: *A*, rectus muscle; *B*, deep epigastric vessels; *C*, external inguinal fossa; *D*, middle inguinal fossa. Observe that the internal inguinal fossa is almost entirely covered by the lower portion of the rectus. (Drawing made from a dissection prepared by Dr. Bonney, Department of General Anatomy, Jefferson Medical College.)

By what force or forces the testicle is made to change its position from abdomen to scrotum has been a subject of considerable discussion. In the light of our present knowledge it may be stated that the conditions operative in bringing about this change are the disparity of growth between the inguinal ligaments and the abdominal wall and the elongation of the vaginal process of peritoneum. Modern embryologists are agreed that the former factor is a very important one. As the abdominal walls develop more rapidly than the inguinal ligaments, there is a tendency for the testicles to be drawn down toward the inguinal region where the ligaments are attached. As this attachment is into the little sac of peritoneum in the genital swellings which develops into the vaginal process, it seems reasonable to suppose that the testicle will be carried still further downward as the process becomes elongated. The theory that the ligament itself draws the testicle downward by contracting is not tenable in view of our present embryological knowledge.

As the testicle passes through the abdominal wall it carries in front of it a layer from each of the muscular or fascial planes which it perforates. Thus, as it comes out of the abdomen at the spot known as the internal ring, it carries a portion of transversalis fascia along, drawing it down like a cone or funnel. Hence the application of the term infundibuliform fascia to this portion of the transversalis fascia. From the next stratum of the belly wall, the internal oblique muscle, it takes up a covering known as the cremaster, and as it passes through the succeeding layer, the aponeurosis of the external oblique muscle, it pushes a fascial investment called the external spermatic fascia in front of it.

The spermatic cord occupies and fills up the channel thus made by the testicle in the substance of the abdominal wall. This channel is known as the inguinal canal. It extends from the internal to the external abdominal ring, and in the adult is about an inch and a half long. It is bounded anteriorly by the aponeurosis of the external oblique muscle and in its outer third by the internal oblique, posteriorly by the transversalia fascia, superiorly by the arched fibers of the internal oblique and transversalis muscles, and inferiorly by Poupart's ligament. A study of hundreds of dissections has convinced us that these structures form the true boundaries of the canal, and that any additional ones which may be given are artificial, inaccurate, and confusing.

It must always be borne in mind that the term "canal" is a misnomer. There was a canal in fœtal life, but under normal conditions there is none in extra-uterine life except for a very short time. In the adult the inguinal canal is nothing but a slight gap in the abdominal wall well filled in by the spermatic cord or round ligament. From the description of the internal abdominal ring it will be readily understood that the term "ring" is also inaccurate (Fig. 3).

The external abdominal ring likewise is not a ring in the true sense of the word. It is a cleft in the aponeurosis of the external oblique muscle, situated just above and external to the spine of the pubes, having its base below and its apex above. It transmits the spermatic cord in the male and the round ligament in the female. The margins of this cleft are called pillars, of which the outer is the stronger, being formed by the inner end of Poupart's ligament. The inner pillar is attached to the symphysis pubis, its fibers interdigitating with those of the internal pillar on the opposite side. Stretching across the ring from one pillar to the other are fibers likewise derived from the aponeurosis of the external oblique, known as the intercolumnar fascia, and continuous with the external spermatic fascia which envelopes the cord. Thus it is seen that there is no distinct opening in the aponeurosis of the external oblique muscle, the cord and intercolumnar fascia effectually closing in the breach between its fasciculi. It is only after the intercolumnar fascia has been removed and the cord displaced that the gap becomes apparent (Figs. 4 and 5).

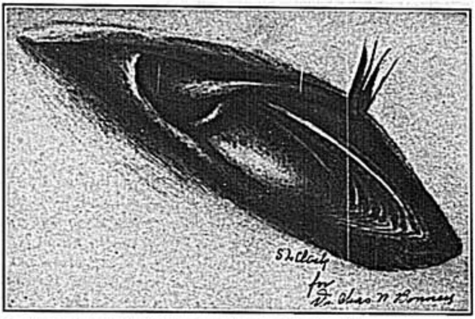

FIG. 3.—The inguinal canal and the internal abdominal ring. The spermatic cord has been lifted out of the canal, and traction is being made upon it so as to render the infundibuliform fascia tense. Observe the deep epigastric artery internal to the ring. (Drawing made from a dissection prepared by Dr. Bonney, Department of General Anatomy, Jefferson Medical College.)

As has previously been stated, a certain number of all inguinal herniæ, certainly not more than four or five out of every hundred, protrude from the abdomen through Hesselbach's triangle. The remainder come out through the internal abdominal ring. They traverse the inguinal canal, break through the external ring, and either present above and external to the pubes or pass downward into the scrotum. A hernia of this kind is known as an indirect, oblique, or external hernia: indirect, in contradistinction to the one which comes out through Hesselbach's triangle; oblique, because of the direction in which it passes downward; external, because it lies to the outer side of the deep epigastric artery. A hernia which passes through the external ring is called a complete inguinal hernia. The coverings of an indirect hernia are derived from the various layers of the abdominal parietes, just as are the coverings of the spermatic cord. From within outward they are peritoneum, preperitoneal fat, infundibuliform fascia, cremaster muscle or fascia, intercolumnar fascia, superficial fascia, and skin. Two points in reference to these coverings are worthy of mention: (1) The peritoneal covering, or sac, of an acquired hernia is derived from the parietal peritoneum, so that when the hernia extends down into the scrotum, its peritoneal covering has nothing in common with the tunica vaginalis testis. This is not true, however, of congenital herniæ, presently to be considered. (2) In enumerating the coverings of a scrotal hernia, the term dartos may be substituted for superficial fascia, the dartos being continuous with the superficial fascia in the lower third of the abdomen, but containing some muscle fibers. Although this point may seem trivial it has often been puzzling to students. Possibly this may be the most appropriate place to state that a scrotal hernia is merely a complete inguinal hernia, either indirect or direct, which has reached the scrotum.

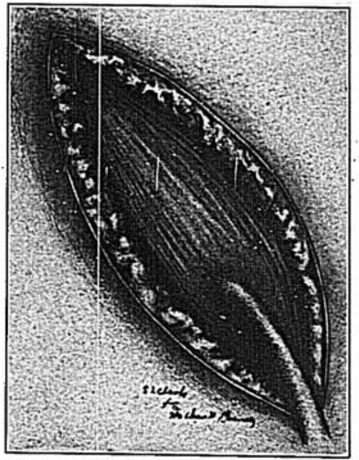

FIG. 4.—Showing the spermatic cord emerging from the external abdominal ring. Observe that there is no distinct opening in the aponeurosis of the external oblique muscle. (Drawing made from a dissection prepared by Dr. Bonney, Department of General Anatomy, Jefferson Medical College.)

A hernia coming through Hesselbach's triangle is called a direct or internal hernia: direct, for the reason that it bulges forward into the lower part of the inguinal canal without following the course of the cord obliquely downward from the internal ring; internal, because it lies to the inner side of the deep epigastric artery. As has already been stated, such a hernia almost always comes out through the middle inguinal fossa. We cannot agree with those writers who state that it more commonly emerges from the internal fossa. The latter is well protected by the rectus muscle in the manner previously described. That a loop of gut may work its way through the unprotected lower external corner of this fossa is not to be doubted, but it is much more likely to come out through the middle fossa, both lateral boundaries of which are less resistant than the strong tough margin of the rectus muscle.

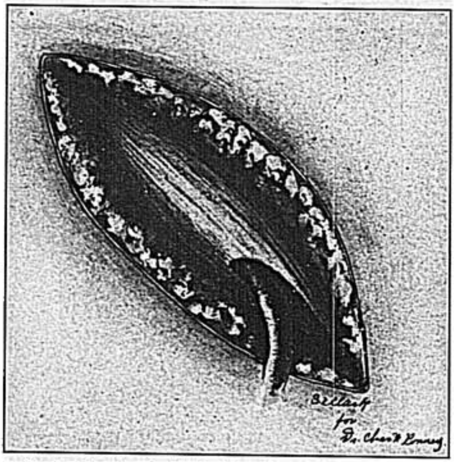

FIG. 5.—Showing the external abdominal ring after the intercolumnar fascia has been removed and the spermatic cord freed. Observe the opening in the aponeurosis of the external oblique muscle. (Drawing made from a dissection prepared by Dr. Bonney, Department of General Anatomy, Jefferson Medical College.)

A hernia escaping through the internal fossa has been termed an internal oblique or vesicopubic hernia. When the sac attains a considerable size it would be difficult to trace its origin unless the urachus could be readily distinguished. It is interesting to note that isolated cases are on record in which a hernia has separated the fibers of the rectus muscle, and even pushed its way between the two recti, presenting at the linea alba.

The coverings of a direct hernia are, from within outward, peritoneum, preperitoneal fat, transversalis fascia, intercolumnar fascia, superficial fascia, and skin. The conjoined tendon is usually enumerated among the coverings of a direct hernia, but, as already stated, it is only in about 25 per cent. of all subjects that such a structure is actually present. When it does exist it is not pushed forward by the hernia, as has generally been taught, but the hernia slips around it, leaving it undisturbed. The tendon, when present, is firmly fixed to the crest of the pubes and iliopectineal line, and above is continuous with the substance of the internal oblique and transversalis muscles. That it could be pushed forward by a hernia is not at all probable, and, moreover, we have never found it displaced in those cases which we have observed at the operating table. In those cases in which there is no conjoined tendon it is possible that some of the attenuated lower fibers of the internal oblique may be carried forward by the hernia, especially if the latter is large. In about forty operations for direct hernia, however, one of us has never observed any muscle fibers in the coverings of the bowel.

It has been frequently stated that about one-fifth of all inguinal herniæ are direct. We are of the opinion that this estimate is much too high. We believe, as we have already stated, that not more than four or five out of every hundred are direct.

Etiology. The causes of inguinal hernia may be considered under three headings: (1) Congenital defects; (2) natural weakness of the abdominal wall in the inguinal triangle, and forces which increase this weakness; and (3) conditions which increase intra-abdominal pressure.

Congenital defects relate chiefly to faulty obliteration of the vaginal process of peritoneum. As previously stated, that portion extending from the internal ring to the top of the testicle usually becomes obliterated between the tenth and twentieth days after birth. The exact manner in which closure takes place is not known. It is generally conceded that obliteration begins in the middle part of the process, and some who have studied the subject also think it commences simultaneously near the internal ring. At all events there are a certain number of individuals in whom closure partly or entirely fails to take place. These persons are particularly predisposed to hernia. When the vaginal process remains open throughout its whole extent there is an uninterrupted passage from the peritoneal cavity to the scrotum into which the intestine can readily find its way, with the result that a hernia is produced. Such a hernia is called congenital, its sac being preformed. The contents of the sac are in contact with the testicle, but the testicle is not suspended by its cord within the sac in the manner that a chandelier is suspended from the ceiling into the middle of a room, although we have found that undergraduates and practitioners of medicine alike sometimes entertain this erroneous conception of the relation of the one to the other. The inguinal portion of the cord lies in the retroperitoneal connective tissue, the scrotal portion is also posterior to the vaginal process, and one layer of peritoneum covers the testicle, namely, the visceral layer of the tunica vaginalis, which is identical with the posterior wall of the hernial sac. Thus, the testicle really lies behind the sac, being separated from the gut by a single layer of peritoneum instead of a double one, as is the case with acquired indirect inguinal hernia. These relations are well illustrated in Fig. 6.

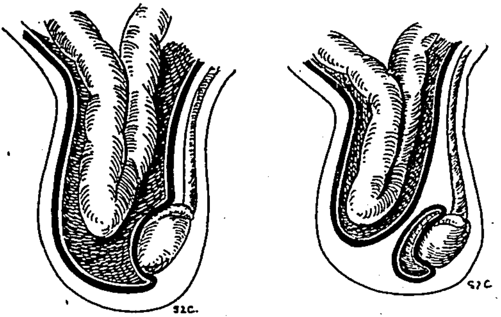

FIG. 6.—The left-hand illustration shows the relation of the testicle to the sac of a congenital scrotal hernia; the right-hand illustration shows the relation of the testicle to the sac of an acquired scrotal hernia. (Adapted from Gray.)

If the vaginal process has become obliterated below, but not above, there will be a channel extending from the abdominal cavity down to the site of obliteration. This channel is called the funicular process, and a projection of intestine into it is known as hernia into the funicular process. This variety differs from congenital hernia only in the extent to which it is possible for the gut to descend.

If the vaginal process is closed above, at or near the internal ring, but remains open below, two kinds of hernia may be formed. In some cases the septum between the abdominal cavity and the unobliterated vaginal process gives way slowly and allows the intestine to pass down behind the vaginal process. Such a hernia is known as an infantile hernia. Its sac is formed by the displaced septum together with the contiguous parietal peritoneum. The bowel has three peritoneal coverings, namely, the two walls of the vaginal process and the true sac of the hernia.

In other cases the septum gives way in such a manner that it and the contiguous parietal peritoneum are carried down into the unobliterated vaginal process. A hernia thus formed is termed an encysted infantile hernia. It has two peritoneal coverings, namely, the anterior wall of the vaginal process and its proper sac.[1]

Finally, a remnant of the vaginal process may persist above at the abdominal ostium, projecting slightly downward into the inguinal canal or appearing merely as a bulging at the internal abdominal ring. That such remnants predispose greatly to the formation of hernia is not to be doubted. In reality they constitute a preformed sac which becomes stretched and elongated by the pressure of the gut which impinges upon them from above. It is no doubt true that a considerable percentage of herniæ which are thought to be acquired originate in these remnants of the vaginal process, and therefore are originally due to a congenital defect. To maintain that all indirect herniæ are dependent upon such a defect, however, as has recently been done by some writers, is to ignore a very important structural condition normally present in the inguinal triangle which may rationally be considered to predispose to the escape of the intra-abdominal contents, namely, the arrangement of the transversalis fascia at the internal ring. This ring, although only a potential one, can be proved both embryologically and anatomically to constitute a weak spot in the parietes; and to deny the possibility and probability of this spot yielding to intra-abdominal pressure is, we think, unwarranted. Another point to be considered is the following: when a mere bulging of peritoneum is found at the internal ring in the adult subject, can it be positively determined whether this bulging represents a remnant of the vaginal process or the beginning of an acquired hernial sac? Certainly not. No one can prove it to be the one, no one can demonstrate it to be the other. The only way to decide the matter beyond question would be to cut a large number of children open in such a manner as to permit an examination for remnants of the vaginal process, sew them up again, and have them watched throughout life for the development of hernia. Until it shall have been determined in this manner that only those persons who possess remnants of the vaginal process develop hernia in adult life, we are unwilling to admit that all indirect inguinal herniæ are of congenital origin. We admit the probability of a considerable percentage being due to the presence of remnants of the vaginal process, but also believe that others develop irrespective of any defect in obliteration of this process.

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[1] We believe that it would be better to abolish the old classification, according to which these herniæ are divided into congenital, infantile, etc., and call them all congenital. One is just as much dependent upon a defect of development as is another, and it certainly would be more accurate, and also probably less confusing to students, to speak of them as different forms of congenital hernia.

Malposition of the testicle is a defect which is frequently associated with inguinal hernia. When the gland is retained high in the inguinal canal it has a tendency to dilate the internal ring and thus facilitate the escape of omentum and bowel. As the bowel passes downward its course may be obstructed by the testicle, so that it rises above the latter, with the result that the sac becomes curved. In cases in which the testicle has passed through the external ring, but has not descended into the lower part of the scrotum, the hernia pushes its way in front of the testicle, but usually does not descend below it. Non-descent of the testicle is also thought to be the chief cause of the rare pre-inguinal hernia, in which the sac escapes from the inguinal canal by pushing its way into the cellular tissue between the aponeurosis of the external oblique muscle and the skin. In this form the sac is divided into two or even three parts—one inguinal, one abdominal, and occasionally also a scrotal portion. The abdominal or superficial sac is the more important. It may be situated between the external abdominal ring and the anterior superior spine of the ilium, just in front of the ring, between the scrotum and the thigh, as in a case reported by Busch; or it may extend up toward the umbilicus, as it did in Broca's case.

Another variety most likely dependent upon a congenital defect is the inguino-interstitial, which develops in the inguinal canal, but works its way into the substance of the abdominal wall instead of emerging through the external ring, which is much smaller than usual or even absent. In the male such herniæ are associated with malposition of the testicle, and the narrowness of the ring was supposed by Tillaux and others to be due to the fact that the testicle had not passed through the external oblique muscle. Some cases, however, have been observed in the female, and, moreover, in others an accessory sac has been found in the scrotum, and the external ring has been observed to be wider than normal. Berger believes that the location of the sac in the substance of the abdominal wall is due to the presence of a congenital diverticulum in the vaginal process of peritoneum, which becomes enlarged by intra-abdominal pressure after the intestine engages in it. Such a hernia usually lies beneath the three lateral muscles of the abdomen, although some have been found between the internal and external oblique, extending high up into the abdominal wall.

Finally, there remains to be described the properitoneal hernia, for the pathogenesis of which a number of explanations have been offered. This variety of hernia also has two sacs, one deep and one superficial. The deep, or properitoneal sac is situated between the peritoneum and the transversalis fascia, and generally extends outward toward the anterior superior spine of the ilium. It may, however, extend downward toward the pubes. The superficial sac occupies the inguinal canal and sometimes extends down into the scrotum, just as any other hernia which follows the cord may do. An angle of varying degree marks the divergence of one sac from the other and there is also a space, forming a vestibule, between them. Although some difference of opinion has existed as to whether properitoneal hernia is congenital or acquired, it has come to be pretty generally admitted that it owes its origin to a congenital defect. Thus, some have taught that the properitoneal sac is formed from a peritoneal diverticulum situated near the inguinal fossæ and that the superficial one is merely a spur carried out from the deep one by a process of dilatation as more and more of the intra-abdominal contents force themselves into it. Others maintain that both sacs are formed from the vaginal process, the deep from the iliac portion, which often presents a retro-inguinal fold well adapted for distension, and the superficial from the remainder of the process. The latter theory is the simpler and, moreover, is the one which those who have recently studied the subject most carefully are inclined to accept.

Perineal ectopy of the testicle may also be associated with hernia. This is infrequent, and is mentioned merely for the sake of completeness.

With regard to direct hernia it may be stated that this variety is never of congenital origin. The middle inguinal fossa is structurally a weak spot, through which the abdominal contents can be pushed by increased pressure from above. It has already been so well described under anatomy that further allusion to its structural peculiarities and relations are unnecessary.

Among conditions which increase the natural weakness of the abdominal wall may be mentioned diseases which impair the nutrition of the muscles, such as rickets, for instance; systemic maladies which produce great emaciation; abdominal tumors which impinge upon the anterior belly wall and distend it; pregnancy, which acts in the same way; accumulation of fat in the omentum and preperitoneal tissue, also tending to stretch the peritoneum; relaxation of the parietes incident to old age; and, finally, repeated efforts in lifting or dragging heavy weights.

It will be readily understood that some of these causes, such as pregnancy and accumulation of fat in the omentum, also increase intra-abdominal pressure. Other potent factors in augmenting pressure are cough, vomiting, and straining to empty the rectum or bladder. Bronchitis, emphysema, asthma, and whooping cough are not uncommonly the immediate exciting cause of hernia. In Berger's series of cases cough was considered an etiological factor more than eight hundred times. Elongation of the mesentery has also been considered a factor in the production of hernia. Although the mesentery is relatively longer in infancy than it is in adult life, it is probably never so short as to prevent the intestines from coming out of the abdomen. This opinion is confirmed by the investigations of Mr. C. B. Lockwood, of London.

Age, sex, and heredity have also to be considered from the standpoint of etiology. In regard to age, it may be stated that hernia is most common in childhood and in middle adult life. Berger, who has studied the age incidence carefully, shows that by far the larger percentage of cases occur in children, that there is a constant decline until the thirtieth year, after which a steady increase takes place until the fifty-fifth year. It should not be forgotten, however, that hernia is relatively frequent in old persons on account of the relaxation of their abdominal wall and their susceptibility to bronchitis, asthma, etc. Were it not for the fact that they lead a shielded life and do not follow laborious occupations, they would be more liable to hernia than those in middle life. Berger's statistics also show that double hernia is rare in infancy and frequent in old age, reaching its maximum at sixty-five years.

All will admit that inguinal hernia is more common in males than it is in females. It is our belief, however, that it is more common in females than it has been thought to be, as the statistics of those hospitals and dispensaries in which female physicians are employed show a greater percentage of cases in women and girls than those institutions where only male physicians are in attendance. In over 800 operations for inguinal hernia which one of us has performed, not more than 50 were upon women.

The role of heredity is not thoroughly understood, but in our opinion it is of unmistakable etiological influence, as we know of many families in which several members have hernia.

PATHOLOGY. In studying the pathology of hernia the sac should receive consideration first. In shape it may be round, cylindrical, conical, or pyriform. In size it varies greatly. That portion just below the internal ring is called the neck, the remaining part the body. It differs in appearance according to the age of the hernia and the complications to which the hernia is subjected, being thin, smooth, and of the color of normal peritoneum in very recent cases; but thick, tough, corrugated, and white, yellow, or grayish in color, much resembling fibrous tissue, in those of long duration. In strangulation, it becomes dark red, purple, or black. In herniæ which have existed for any length of time it becomes blended with the other coverings of the bowel, frequently to such an extent that its separation is very difficult or even impossible. This is particularly true in regard to its relation with the transversalis fascia. In old herniæ there has usually been sufficient inflammation to blend the two layers inseparably together. One of us has had the opportunity of making postmortem dissection of a number of large scrotal herniæ, evidently of many years' duration, and has found that it is impossible to separate the coverings into more than two layers after the herniated mass has been freed from the scrotum. One of these layers has invariably been traced to the aponeurosis of the external oblique muscle, the other has always disappeared within the internal hernial orifice. It is our opinion that the internal layer is composed of both peritoneum and transversalis fascia. In no case has it been possible to find any traces of cremaster muscle fibers. The external layer has always been adherent to the internal surface of the scrotum.

As the result of inflammation, which is bound to occur in the course of time, adhesions form which prevent the reduction of the sac with its contents. In the dissections just mentioned, the sac, together with other coverings, has been firmly attached to the pillars of the external ring, so that considerable cutting was necessary to free it.

Adhesions also not uncommonly form between the sac and its contents, a circumstance which still further renders reduction difficult or imperfect. Some of these inflammed areas may undergo calcareous change. Finally the inflammatory process may cause partial obliteration of the sac. It commences at the neck and may be confined to this part, but in some cases it extends downward and causes a variable degree of contraction. If obliteration at the neck be complete, a cyst may be formed in the lower part of the sac, owing to retention of the secretion which is produced. If a zone of obliteration occurs below the neck, the sac will be divided into two parts. These multiple sacs have been found both in congenital and acquired hernia. Longitudinal fibrous bands have also been observed. We have seen a few of these ourselves, and they have been mentioned by Okinczyc. No doubt others have also noticed them.

Hernia always produces changes in the abdominal parietes, the alterations depending upon the size of the hernia. The inguinal canal is always dilated, and the two abdominal rings are stretched and more or less approximated. The deep epigastric vessels may be displaced inward. Large herniæ cause pressure atrophy of the lower fibers of the abdominal muscles, and when they descend into the scrotum may likewise produce atrophy of the testicle. The extent to which the inguinal region may be weakened is well illustrated by the conditions which one of us recently observed in the case of a dissecting room subject who presented a large irreducible scrotal hernia which showed every evidence of having existed for years. The lateral muscles on the affected side in the lower part of the abdomen had become converted into a thin homogeneous layer, looking much like a piece of sheepskin; the aponeurosis of the external oblique had lost its firmness, presenting a number of small loose folds for a considerable distance above the anterior superior spine of the ilium, and the lower margin of the external ring had been pushed down below Poupart's ligament almost to the saphenous opening. There was hardly any space between the superior margin of this greatly dilated external ring and the internal hernial orifice.

In reference to the contents of inguinal hernia, it may be stated that they are usually composed of omentum and small intestine. The large intestine, the stomach, the uterus and ovaries, the bladder, in fact any of the intra-abdominal contents but the pancreas may occasionally escape through the inguinal region. It is stated that a floating kidney has been found in the sac of an inguinal hernia.

The contents are subject to morbid changes. Thus the omentum is never normal except in very recent cases. Its adherence to the sac has already been mentioned. Its folds also become adherent to one another and sometimes portions of it become attached to the intestine. At the neck of the hernia the omentum is also often found converted into a fibrous band, this condition being the result of inflammation.

From a practical point of view the most important change which takes place in hernia is strangulation, by which is meant an occlusion of the lumen of the intestine together with arrest of its blood supply, produced by a constriction, which also renders the hernia irreducible if it was not already so before the said constriction occurred. As a result of strangulation the passage of both feces and gas is stopped and gangrene supervenes in the herniated bowel.

The most common site of strangulation in inguinal hernia we believe to be at the external abdominal ring, and not at the internal ring or within the sac, as has frequently been taught. Certainly this has been our experience, and we believe also that of most modern surgeons. In recent herniæ it may occur in the inguinal canal, between the two rings, being due probably to inflammatory changes in the transversalis fascia. In congenital hernia strangulation may be produced by strands or valves within the sac, or by the neck of the sac itself. In like manner it may result from the action of the inflammatory bands present in a partly obliterated sac, or be caused by the omentum, which, becoming adherent to the sac, forms a band that constricts the intestine. Rarely the bowel has perforated the omentum and become strangulated. Finally the possibility of torsion of the mesentery within the hernial sac must be borne in mind as a possible cause of strangulation.

The appearance of the strangulated intestine depends upon the length of time which the constriction has lasted. At first deep red, it gradually becomes darker and darker, until it may be almost or quite black. Its lustre disappears, patches of fibrinous deposit become visible, and here and there breaches in the continuity of the serous covering take place. These changes are the forerunners of gangrene, being shortly superseded by sloughs, which in turn may lead to perforation of the bowel. If the strangulation be relieved, the constriction of the intestine is plainly discernible.

The sac of a strangulated hernia is usually round in shape and almost always contains a considerable quantity of fluid. The sac, like the intestine, is much congested and may even share in the gangrenous process with which the latter becomes affected. We have often found the sac of a strangulated hernia to be of a dull slate color, with violaceous or dark brown patches scattered over its surface. The liquid in the sac is at first thin, clear, and of a light yellow color; but it soon becomes thick, turbid, and of a reddish or brown tinge, and contains flakes of fibrin. It may soon assume a fecal odor, caused by the presence of the colon bacillus, which migrates easily through the walls of the damaged bowel. Occasionally there is no fluid, the bowel being in direct contact with the internal surface of the sac. It is important to remember this fact and not to depend upon an outflow of fluid as a necessary sign that the bowel has been reached. Rarely it happens that only a segment of the bowel becomes constricted. To this condition the term lateral strangulation may be applied (Richter's hernia). Although the caliber of the bowel is diminished, it may nevertheless be possible for gas and fecal matter to work their way through. The bowel immediately below the constriction is usually found dilated, so that it may present the appearance of a diverticulum.

By an obstructed hernia is meant one in which the intestinal contents have accumulated to such an extent as to block the fecal current. The term incarcerated hernia has also been applied to this condition, but we believe it to be an inaccurate one, as it does not convey the idea of the condition which actually exists. If the term incarcerated is to be used at all, we believe it should be applied to those herniæ which are temporarily irreducible. We insist that this would be the correct use of the term. Obstructed hernia is probably much rarer than it was formerly supposed to be. Its existence has even been denied, but without sufficient reason, we are sure. We have seen the condition a number of times, and Nau has recently collected 2[illegible] authentic cases. It occurs almost always in herniæ of the large intestine, which have become irreducible as the result of inflammation. Intestine thus imprisoned loses its peristaltic power more or less, and this functional impairment, together with the mechanical conditions present, permit the occurrence of a gradual fecal obstruction.

A hernia may be attacked by tuberculosis or malignant disease. Cotte[2] has recently collected 136 cases of tuberculosis—the most thorough study of the subject with which we are familiar. The sac of the hernia alone, or both sac and contents, may be affected. Jonnesco states that the intestine may be diseased and the sac healthy, but his statement has not been generally accepted. In children the sac is usually the site of the tuberculosis; in adults both sac and contents are usually affected. Three forms of tuberculosis have been observed in the sac, namely, the miliary, the caseous, and the fibrous. Of these, the miliary is the most common. It is evidently the same lesion as that of miliary tuberculous peritonitis. There is generally considerable fluid in the hernial sac. Next in order of frequency is the caseous form, the fibrous being the rarest of all. The lesions of the intestines are no different from those found when the bowel becomes tuberculous in its normal habitat.

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[2] Revue de Gynecologie, November and December, 1906.

The neoplasms which have been found associated with hernia are carcinoma, sarcoma, and lipoma. They may affect the sac, the intestine, the omentum, or whatever organ is contained within the sac. The lipomas naturally develop in the omentum.

In another paper the diagnosis and treatment of inguinal hernia will be discussed.

THE ACTION OF THE SHORT ROTATORS ON THE NORMAL ABDUCTION OF THE ARM, WITH A CONSIDERATION OF THEIR ACTION IN SOME CASES OF SUBACROMIAL BURSITIS AND ALLIED CONDITIONS.

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Codman, of Boston, in a recent monograph on lesions of the shoulder-joint due, in his opinion, to trauma or to primary inflammation of the subacromial bursa, has done more to clear up the interesting subject of painful shoulders, and to explain certain definite restrictions of motion between the humerus and the scapula than has been accomplished by any other investigator, and his masterly interpretation of the mechanics of the shoulder-joint is so simple, clear, and conclusive (as far as he goes) as to establish almost beyond question his contention as to the primary action of the supraspinatus in abduction of the humerus preliminary to the action of the deltoid, which is thus permitted to come into action and exert its force in the proper direction to complete the arc of abduction. He has followed his theory through the operating room and into the dissecting room, and has conclusively established his contention by anatomical specimens showing the pathological changes following rupture of the supraspinatus, with the subsequent loss of the power of abduction in these cases. He has applied his researches into the normal mechanics of the scapulohumeral joint to the practical treatment of a class of shoulder lesions showing restriction of motion and tenderness below the acromion, which, as he says, constitute by far the largest class of patients applying to any surgical clinic for the relief of trouble referable to the shoulder-joint. For this class of cases he has adopted Kuster's term of subacromial bursitis in place of the subdeltoid bursitis used by him in his first paper, although the latter term would seem to be by far the better, since more of the bursa is subdeltoid than subacromial, and since Piersol, in his recent anatomy, used the term subdeltoid in his description of the bursa. Whatever term is used, the credit must go to Codman for the most convincing description of the mechanics of this complicated joint. And in the light of his investigation the real action of the joint does not appear to be so complicated after all. A study of normal shoulder action is necessary for the proper understanding of his cases, and the few which have come under observation which we hope to demonstrate are departures from the regular types which he has described. If one considers anatomically the origin and insertion of the various muscles surrounding the shoulder-joint, and if one bears in mind the fact that the real capsule of the joint is formed by these surrounding muscles, and not by what is known as the capsule, with its ligamentous bands, which is a lax structure neither aiding nor opposing the normal motions of the joint, it would seem to be easy to assume, and equally easy to prove from a study of the anatomical structure, that the first few degrees of motion in abduction are due to the supraspinatus contraction, and is preliminary or preparatory, as it were, to the action of the deltoid.

Piersol says that the capsule of the shoulder-joint, among other uses, by means of its tense, firm under surface helps to prevent the arm from being raised beyond a certain point in abduction, but it is extremely doubtful if the capsule has any such action, although this one is by far the more probable of all the uses ascribed to it by the various anatomists, and it is probable that the capsule per se has very little retarding action on the movements of the joint itself. As Codman says, it is necessary to disregard our previous teaching and conception of the structure which has been called the capsule. The real capsule of the joint is made up of the muscles which surround it. Codman's contentions, which are, I think, accepted for the most part or will be so accepted by all who carefully examine the anatomical structure of the shoulder-joint, bearing in mind the laxity of the capsule, are, briefly, that the first few degrees of motion in abduction of the humerus are always due to the contraction of the supraspinatus acting on the short arm of the lever, the fulcrum of which is on the glenoid, the long arm of the lever extending downward through the arm (Fig. 1).

It will be readily seen that the fulcrum of such a lever must be a constantly changing point, and that the action of such a lever would be comparatively slight, and such seems to be the case. From its insertion high up on the greater tuberosity of the humerus the action of the supraspinatus must be limited in power, because the power in that case would be applied so near the fulcrum, while the weight